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# *Streptobacillus moniliformis* as the Causative Agent in Spondylodiscitis and Psoas Abscess after Rooster Scratches▽

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**We report a case of *Streptobacillus moniliformis* spondylodiscitis accompanied by a psoas abscess in an 80-year-old man scratched by a rooster. *S. moniliformis* was identified from abscess fluid by use of 16S rRNA gene sequencing. After 18 weeks of antimicrobial therapy, the clinical condition of the patient improved.**

## CASE REPORT

In 2005, an 80-year-old farmer was admitted to the university hospital of Clermont-Ferrand, France, for treatment of deterioration of his general health and inflammatory syndrome. In 1993, he had undergone total left hip replacement; in 2000, his right hip was also replaced. At the time of his admission, he was undergoing no treatment and had no known allergy. His symptoms had begun 1 week earlier, a few days after a rooster had scratched his left hand; he experienced shaking chills without fever and back pain that irradiated to both legs on awakening. The pain gradually disappeared.

On admission, physical examination showed no specific signs. Laboratory investigations revealed functional renal insufficiency. His leukocyte count was $19 \times 10^9$/liter, his neutrophil count was $18 \times 10^9$/liter, the serum level of C-reactive protein (CRP) was 488 mg/liter (a normal level is <5 mg/liter), the serum level of fibrinogen was 8.9 g/liter (a normal level is 2 to 4 g/liter), and the level of procalcitonine was 13 ng/ml (a normal level is <0.5 ng/ml). Four blood specimens were inoculated into the Bactec blood culture system (Becton Dickinson Diagnostic Instrument Systems, Le Pont-De-Claix, France) (paired aerobic and anaerobic bottles). Two anaerobic bottles gave positive results after 1 day of incubation, and one anaerobic bottle gave positive results after 5 days. Two aerobic bottles gave positive results after 3 and 2.5 days of incubation. Gram-stained smears of the bottle contents showed pleomorphic forms with fusiform gram-negative rods. Contents of bottles that gave positive results were plated on Columbia sheep blood agar (Oxoid) and incubated under aerobic and anaerobic conditions at 37°C. Cultures were obtained only under anaerobic conditions, but subcultures gave negative results, thereby preventing identification by conventional biochemical and carbohydrate analysis. Attempts to identify the bacterium by 16S rRNA sequencing from agar cultures failed. Empirical antibiotic therapy with parenteral amoxicillin-clavulanic acid (1 g) every 8 h and ofloxacin (200 mg) every 12 h was started. An abdominal computed tomography scan revealed a right iliac psoas abscess in contact with a screw of the right prosthesis. Aspiration of the abscess was performed under ultrasound guidance. Gram-stained smear results showed pleomorphic fusiform gram-negative rods. Abscess fluid was plated on Columbia sheep blood agar and incubated under aerobic and anaerobic conditions at 37°C for 48 h. Cultures were negative for bacterial growth.

The diagnosis was made by use of a direct 16S rRNA PCR assay with fluid from the abscess. Primers 5′-TCAAAGTGA ATTGACGGGGGC-3′ and 5′-GCCCGGGAACGTATTCA C-3′ were used to amplify the conserved 16S rRNA genes from the psoas abscess, and a 450-kb amplicon was obtained. Comparison with sequences deposited in the GenBank database showed that a partial sequence (the region consisting of positions 887 to 1336) shared 99% homology with that of the 16S rRNA gene of *Streptobacillus moniliformis* (accession numbers AB330757 and DQ325537).

The patient was transferred to the orthopedic department. His CRP level was 240 mg/liter. His sepsis status had been worsening, and he presented with disorders of consciousness. A thoracic, abdominal, and brain computed tomography scan revealed pericardial and pleural effusions and collections in the right iliac psoas. Effusions and collections were drained. All the culture results were negative. Antibiotic therapy was switched to imipenem-cilastatin (1 g) every 12 h, ciprofloxacin (400 mg) every 12 h, and teicoplanin (600 mg) once a day. His CRP level decreased to 163 mg/liter. One week later, the patient became febrile, with evening peaks in temperature (39°C). Blood culture results remained negative. He was transferred to the department of infectious diseases. His general condition was severely deteriorating, with back pain and loss of weight. Laboratory investigations showed a CRP level of 115 mg/liter and a fibrinogen level of 7 g/liter. A bone scan indicated an increased signal at L3. An additional 9-week treatment with intravenous antibiotics, including ofloxacin (200 mg) every 12 h, clindamycin (600 mg) every 8 h, and metronidazole (500 mg) every 8 h, was started. His clinical condition improved, and 1 month later his CRP level was 4 mg/liter. A magnetic resonance imaging scan of the lumbar spine confirmed the diag-

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nosis of psoas abscess and of spondylodiscitis at T5 and T6 and, in particular, at L2 and L3. Eight months after the end of the antibiotic treatment, the patient was in good health.

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*S. moniliformis* as the causative agent of spondylodiscitis and psoas abscess is an uncommon finding. To our knowledge, this is the first reported case involving this bacterium. There is one documented case of infection with *S. moniliformis* that manifested as acute polyarthritis with involvement of the spine following ingestion of milk (Haverhill fever) (1). *Staphylococcus aureus* remains the most commonly identified etiologic agent in cases of spondylodiscitis. However, a wide variety of other etiologic agents have been incriminated, such as *Nocardia nova* (7) and *Kingella kingae* (2).

*S. moniliformis* is the causative agent of rat bite fever (10), and between 50 and 100% of rats are colonized in the nasopharynx (5). The disease is most often transmitted from the bite of an infected rodent. There have been reports of infection or colonization in mice, guinea pigs, gerbils, ferrets, cats, and dogs (4). After exposure, the incubation period ranges from 3 days to over 3 weeks but typically is less than 7 days (4), as in our case. Our observation is the first to be associated with a rooster scratch. In the absence of direct contact with rats during the incubation period, the rooster seemed to be the best candidate for the origin of infection in our patient. However, no confirmatory evidence exists to prove the risk of transmission from this animal.

Rat bite fever is a systemic disease, typically presenting with fever and rigor, skin rash, and migratory polyarthralgias. In our patient, only shaking chills and arthalgia were noted. Serious complications, such as myocarditis, pericarditis, meningitis, amnionitis, polyarthritis affecting the small and large joints, and abscesses in a variety of organs, have also been reported (3, 4, 8). In our observation, the bacterium infected first the spinal column and then the psoas muscle, which was in contact with a screw of the right prosthesis, which caused the inflammatory syndrome.

Efforts at diagnosis based on bacterial cultures from blood were unsuccessful, but Gram staining results, which showed the presence of pleomorphic fusiform gram-negative rods, were evocative of *S. moniliformis*. This bacterium is an extremely fastidious organism whose growth requires microaerophilic conditions and special media such as Trypticase soy agar or broth enriched with 20% blood, serum, or ascitic fluid (4). After antibiotic treatment, molecular techniques were required to make the diagnosis using fluid from the abscess. These techniques were utilized because of difficulties in identifying this bacterium.

Penicillin is the treatment of choice for proven or highly suspected cases of rat bite fever (4). For our patient, diagnosis was considerably delayed, owing to poor recognition of the clinical features and the failure of microbiological diagnosis. As a result, he first received amoxicillin-clavulanic acid and ofloxacin, followed by imipenem-cilastatin, ciprofloxacin and teicoplanin; treatment ended with ofloxacin, clindamycin, and metronidazole. Tests of *S. moniliformis* antibiotic susceptibility by the disk diffusion method usually demonstrate sensitivity to penicillins, carbapenems, clindamycin, and teicoplanin but intermediate sensitivity to fluoroquinolones (9, 12). In our patient, the development of spondylodiscitis may have been due to the use of nonspecific antimicrobial therapy or to an inadequate penicillin dosage. This highlights the importance of appropriate diagnosis. However, even with the right treatment, when the clinical response to antibiotics alone is suboptimal (for example, because of poor antibiotic penetration into the abscess), surgical intervention is required for local drainage to remove any infections and to reduce the bacterial load in the abscess (11). Several drainages were necessary in our patient to enhance treatment.

*S. moniliformis* infections are still difficult to diagnose, especially when clinical signs are not specific and the animal vector is not a rat. Ensuing complications, such as the spondylodiscitis that occurred in our patient, are serious. *S. moniliformis* infection should be suspected when there is contact with an animal and when the bacterium proves to be a fastidious organism. Rats are becoming increasingly popular as pets, and there is evidence that rat bite infections are an emerging syndrome (6).

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